Human Electro-Muscular Incapacitation (HEMI) Devices Characterization: A Comparative Study on Stress and the Physiological Effects on Swine

J.R. Werner[t] , D.M. Jenkins[*], W.B. Murray[^^],E.L. Hughes[*], D.A. Bienus[t], and M.J. Kennett[^t]

[*]Applied Research Laboratory, The Pennsylvania State University, University Park, PA 16802

[^t]Department of Veterinary and Biomedical Sciences, College of Agricultural Sciences, The Pennsylvania State University, University Park, PA 16802

[t]Animal Resource Program, The Pennsylvania State University, University Park, PA 16802

[^^]Milton S. Hershey Medical Center, The Pennsylvania State University, Hershey, PA 17033

Corresponding author: J R Werner

101 Centralized Biological Laboratory

University Park, PA 16802

jrw140@psu.eduTelephone: 814-865-1495

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14. ABSTRACT

Human Elecor-Muscular [sic] Incapacitation (HEMI) devices or Electro-muscular Disruption (EMD) devices are increasingly used in police and military applications. Most individuals who experience electro-muscular incapacitation are in a stress-filled state, and the effets of prolonged or repeated exposures are not well understood. Three different commercially available EMD devices were tested randomly on six anesthetized pigs each for a total of eighteen pigs. Each animal was exposed to an initial 60 second application of the EMD device as an initial stressor. The animals were then allowed to rest under anesthesia for 60 minutes followed immediately by a 180 second application of the same device. Arterial blood gasses and serum samples were collected throughout the experiment to measure catecholamines (epinephrine, norepinephrine, and dopamine) and cortisol. All devices produced some level of muscle tetany as a result of the electrical delivery to the animal. All pigs showed a mixed metabolic and respiratory acidosis. Cortisol tended to decrease after the initial exposure and slightly increased over the rest period. The extreme muscular work caused by the electrical stimulation resulting in muscle contractions did not result in a strong stress response but did result in an immediate sympathetic response during both applications of the device leading to the conclusion that initial stressor followed by rest and prolonged EMD device application did not exhaust the sympathetic system. For healthy adult animals, despite the prolonged muscular exertion and physiological stress caused by EMD devices, the body should be able to mount an appropriate sympathetic response and recover normally.

15. SUBJECT TERMS
non-lethal weapons, catecholamines, cortisol, long duration

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Human Electro-Muscular Incapacitation (HEMI) Devices Characterization: A Comparative Study on Stress and the Physiological Effects on Swine

Abstract

Human Electro-Muscular Incapacitation (HEMI) devices are increasingly used in police and military applications. Typical applications require short exposures; however, there may be need in certain circumstances to apply prolonged exposure of these devices to detain an individual. Most individuals who experience electro-muscular incapacitation are in a stress-filled state, and the effects of prolonged or repeated exposures are not well understood. Three different commercially available HEMI devices were tested randomly on six anesthetized pigs each for a total of eighteen pigs. Each animal was exposed to an initial 60 second application of the HEMI device as an initial stressor. The animals were then allowed to rest under anesthesia for 60 minutes followed immediately by a 180 second application of the same device. Arterial blood gases and serum samples were collected throughout the experiment to measure catecholamines (epinephrine, norepinephrine, and dopamine) and cortisol. All devices produced some level of muscle tetany as a result of the electrical delivery to the animal. All pigs showed a mixed metabolic and respiratory acidosis. Cortisol tended to decrease after the initial exposure and slightly increased over the rest period. The extreme muscular work caused by the electrical stimulation resulting in muscle contractions did not result in a strong stress response but did result in an immediate sympathetic response during both applications of the device leading to the conclusion that initial stressor followed by rest and prolonged HEMI device application did not exhaust the sympathetic system. For healthy adult animals, despite the prolonged muscular exertion and physiological stress caused by HEMI devices, the body should be able to mount an appropriate sympathetic response and recover normally.

Key words

Nonlethal weapons, catecholamines, cortisol, long duration

INTRODUCTION

Human Electro-Muscular Incapacitation (HEMI) Devices are non-lethal weapons that are increasingly used in stressful and complex police and military applications with either repeated 5 second or sustained long duration exposures (30 seconds to 3 minutes or longer). HEMI devices produce low electrical current with high frequencies that when applied to a subject renders them incapacitated by causing muscle convulsions and temporary extreme muscle tetany. Multiple devices exist in the market for military, law enforcement and civilian use. Each device provides different methods of delivering electrical current to the subject resulting in different effects and ability to incapacitate (Hughes et al. 2010).

Incapacitation of an exposed subject occurs only as long as the device is engaged. Typical law enforcement applications of these devices require only short engagements (five second application to subdue the subject followed by subsequent five second applications as deemed necessary to safely place a subject in custody).

Military applications of HEMI technology may require longer exposures than typical law enforcement applications. Differentiating between combatants and noncombatants continues to be one of the significant complexities that our military forces face during counterinsurgency operations. Soldiers are confronted with similar challenges when they are deployed domestically in support of disaster recovery and humanitarian relief efforts. Escalation-of-Force (EoF) procedures have been designed to prepare soldiers to apply necessity, proportionality, and reasonableness in the proper use of force. The application of non-lethal technologies, in particular, the use of HEMI devices with long duration exposures, may allow military forces the means by which they might properly implement EoF procedures and establish control of adverse situations to achieve positive outcomes for all parties, particularly innocent bystanders. They

provide flexibility by allowing soldiers to apply a measure of force with reduced risk of serious injury or fatalities, but still in such a manner as to provide protection of the public and effect compliance. To ensure these non-lethal devices achieve a repeatable human effect and at the same time do not reach a threshold of serious injury of death, the Department of Defense conducts a rigorous testing regimen that includes human surrogate testing to gain insight into these effects and their thresholds. This is an important component of the formal acquisition process.

Since individuals who experience electro-muscular incapacitation are often in a stress-filled state (e.g. running to evade capture, a combat situation, or under the influence of alcohol and/or drugs) it is important to determine if the physiological effects of HEMI exposure differ in unstressed and stressed individuals. In addition, the effects of prolonged or repeated HEMI exposures on individuals are not well understood. The purpose of this article is to report the findings of a study that compared the physiological effects of three HEMI devices in a pre- and post-stressed, anesthetized swine model. The results of this study will assist in the development of standard operating procedures and safety measures to be used when employing these devices.

The three HEMI devices tested in this study were the TASER®X26, TASER®C2 and Stinger™S-200. In a previous study conducted by this group (data unpublished), neither metabolic acidosis nor cardiac arrhythmias were the cause of death in tested swine. Two working hypotheses arose: (1) the subjects are undergoing a prior high level of sympathetic tone, and the HEMI application would over-stress the subject, leading to death or (2) the subjects have exhausted the reserves of their sympathetic systems, and subjects fail to produce additional sympathetic output to support the heart and cardiovascular system (too little sympathetic output).

Catecholamines (markers of sympathetic output) and cortisol were measured after long duration HEMI exposure in stressed and unstressed conditions to test these hypotheses.

Methods

EXPERIMENTAL APPROACH TO THE PROBLEM

To study the hypotheses, three HEMI devices were randomly assigned to each animal, and each device was used on six separate anesthetized animals. The first application of the device was for 60 seconds. The length of this exposure period was selected to be consistent with the possible operational use of all three devices. After a 60 minute rest period (during which the animal remained anesthetized), the animal was exposed to a second 180 second application of the same device. The first 60 second exposure served as a stressor for the second exposure. The 60 minute resting interval was designed to allow the acute phase responses to dissipate while leaving some level of residual sympathetic stress. The second 180 second exposure was intended to provide a maximally stressful event in an animal that was partially recovered from the first exposure. The sequential application served as a model for field conditions where humans are exposed to these devices under stressful conditions (physiological stress resulting from physical, mental, or pharmacological sources).

SUBJECTS

Eighteen healthy domestic swine (*Sus scrofa*) three to five months in age and weighing between 45-75 kg were used for this study. All animal experiments were reviewed and approved by The Pennsylvania State University Institutional Animal Care and Use Committee and the United States Department of Defense Director of Veterinary Affairs and complied with the current laws of the United States of America. Swine are commonly used as models for a variety

of human diseases and conditions including previous HEMI studies. Swine were selected due to relevant similarities in size, cardiovascular and epithelial systems, and general mammalian physiology. The cardiovascular system of the swine model closely resembles that of a human, and is therefore used extensively for testing involving the cardiovascular system. In addition, the sizes of the swine were within the range of human size and weight allowing minimization of the scaling problems (e.g. voltage and amperage) of the HEMI devices.

PROCEDURES

Swine were given a preanesthetic sedative of 1 mg/kg sodium xylazine (Xyla-Ject®, Phoenix, St. Joseph, MO, USA) mixed in a syringe with 6-10 mg/kg ketamine hydrochloride (Ketaject®, Phoenix, St. Joseph, MO, USA) intramuscularly. A 22Gx1" catheter was inserted into the auricular vein, and equal amounts of 100 mg/ml sodium xylazine and 100 mg/ml ketamine hydrochloride mixed in a syringe were administered intravenously to induce anesthesia. Swine were intubated and placed on 100% oxygen plus isoflurane (Isoflurane, USP, Phoenix, St. Joseph, MO, USA) and were mechanically ventilated (Veterinary Anesthesia Ventilator, Hallowell EMC, Pittsfield, MA, USA) throughout the experiment. Using a portable patient monitoring device (Advisor® Vital Signs Monitor, SurgiVet Patient Monitoring, Smiths Medical PM, Inc., Waukesha, WI, USA), swine were instrumented to monitor physiological parameters throughout the experimental procedures. A pulse oximeter with a plethysmogram wave form was attached to the animal's ear and linked to a computerized unit to monitor pulse rate and oxygen saturation (SPO_2). A capnograph measured end tidal CO_2, and swine were fitted with a five lead electro-cardiogram (EKG). Arterial catheters were placed in a branch of the femoral artery to allow the measurement of arterial blood pressure, ensure a reliable monitor of regular heart beat, and enable arterial blood gas sampling.

Animals were mechanically ventilated after intubation. The tidal volume was set at fifty percent (50%) above the estimated normal resting tidal volume with. Swine were maintained at a surgical plane of anesthesia with mechanical ventilation of 100% O_2 and isoflurane up to the point of electrical incapacitation device stimulation. Immediately prior to device stimulation, isoflurane gas was turned off, swine were administered equal parts of 100 mg/ml sodium xylazine and 100 mg/ml ketamine hydrochloride intravenously to maintain surgical plane of anesthesia through device application, and they were ventilated with 100% oxygen. Immediately following device application, anesthesia was continued with isoflurane.

Swine were placed in right lateral recumbency. Each HEMI device had two probes which must contact the animal to complete a circuit to allow electrical current to pass to the animal. One probe was placed in the left axillary area while the second probe was placed in the right inguinal area for HEMI exposures. Each HEMI device was continuously activated for one minute. After a 60 minute waiting period with the animal under anesthesia but with no HEMI device application, the device was reactivated for three continuous minutes. Six arterial blood samples per animal were drawn over the course of each experiment. A baseline sample was drawn after the animal was anesthetized and stable but prior to HEMI device application. A second sample was drawn immediately after the one minute device application. The third, fourth, and fifth samples were taken prior to, during (1.5 minutes into), and immediately following (three minutes) the second exposure. A sixth sample was taken five minutes after the second exposure. If an animal expired, a blood sample was taken immediately following death. Cortisol and catecholamines were measured at all six time points, and blood gases with electrolytes were evaluated for the first five time points. Heparinized blood samples were placed on ice and immediately processed (blood gases and electrolytes). The heparinized blood was

spun in a centrifuge to collect plasma, and the plasma was stored at -40°C until catecholamine analysis could be conducted. Clotted blood samples were centrifuged to collect serum for cortisol analysis.

Blood gases and electrolytes were obtained by processing samples using an *i-STAT* handheld clinical analyzer (HESKA Corporation, Loveland, CO). Serum was sent to a commercial laboratory (ANTECH, Diagnostics, Irvine, CA) for cortisol measurements using immunoassay according to good laboratory practice (GLP) procedures. Catecholamine levels were obtained from analysis of plasma samples at the University of Connecticut. Free swine plasma catecholamines were quantified first by absorbing the catecholamines onto alumina, washing, and eluting with a dilute acid, and then by analyzing using High Performance Liquid Chromatography (HPLC) with electrochemical detection. Extractions were performed using an extraction kit (*ESA Plasma* Catecholamine kit) (*ESA Biosciences*, Chemsford, MA) which included a solid-phase extraction method following modified alumina extraction methods first described by Anton and Sayre (1962), and samples were transferred into auto sampler vials for analysis. The mobile phase was *Cat-A-PhaseII® Mobile Phase* (*ESA Biosciences*, Chelmsford, MA) containing methanol, acetonitrile, phosphate buffer, and an ion pairing agent). The mobile phase was pumped isocratically at 1.0 ml ·min-1. As stationary phase an analytical column (*ESA Catecholamine HR-80*)(8 cm x 4.6 mm ID column packed with 3 micron c18 stationary phase) (*ESA Biosciences*, Chelmsford, MA) was used, with a guard column (C18 MG, 3 micron, 10mmx4.0mm) (*ESA Biosciences*, Chelmsford, MA). The analytical and conditioning cells were optimized for voltage changes prior to analysis. An injection volume 50-µl per sample was injected via an automatic sampler (*Beckman Coulter System Gold 508 Autosampler*, Fullerton, CA). All samples were run in duplicate. Standard solutions of norepinephrine, epinephrine,

dopamine and dihydroxibenzilamine (internal standard) were prepared daily and injected for the calibration curve. Retention times (min) [meanstandard deviation (s.d.)] were: norepinephrine=5.370.43; epinepthrine=6.430.49; dihydroxibenzilamine=10.530.67; dopamine=13.850.85. Quantification of norepinephrine, epinephrine, and dopamine in plasma samples were performed using dihydroxibenzilamine as internal standard.

For purposes of this experiment, death was defined as cardiac arrest plus 30 seconds to ensure no spontaneous return of cardiac action. Cardiac arrest was defined as no effective output from the heart as detected by zero pulse oximeter waveform, zero arterial pressure waveform, and zero heart sounds. This ensured that electro-mechanical dissociation (also called pulseless electrical activity or PEA) was included in the definition of cardiac arrest. Pulse, heart rate, and arterial blood pressure were monitored to indicate when cardiac arrest occurred. Pulse and heart rate were monitored using both electronic and non-electronic means (i.e. direct auscultation). Arterial blood pressure was monitored electronically by storing the serial output from the SurgiVet Advisor Animal Monitor (Smiths Medical PM, Inc., Waukesha, WI, USA) on a computer using the program Hyper Terminal (Microsoft Corp. Seattle, WA.)Core body temperatures were measured by rectal thermometer at several experimental time points.

STATISTICAL ANALYSIS

Data were entered into a Microsoft Excel spreadsheet, and data entry checked. Accuracy was further confirmed by ensuring maximum and minimum data points were within physiological acceptable ranges. Averages, standard deviations, standard errors, counts, maximums and minimums were calculated by programming the spreadsheet. Statistically significant differences between groups were sought using Student's t-test for parametric data that was normally distributed. A Chi square test was used for parametric data which was not

normally distributed, as well as for non-parametric data. When expected cells were less than 5, the Chi square test becomes unreliable, and a Fisher Exact Probability Test was employed. A P-value of less than 0.05 was considered significant.

RESULTS

All HEMI devices produced some level of muscle tetany as a result of the electrical delivery to the animal. The Stinger™S-200 and TASER®X26 both resulted in tetany (sustained muscle contraction). The cyclic changes and intermittent low pulse repetition rate (8pps and 12pps) of the TASER®C2 resulted in observable severe and violent intermittent muscular responses by the animals. During the three-minute exposure, animals generally were observed to experience less muscle stiffness after 60-90 seconds of exposure. At this same time point, diaphragmatic breathing was observed. Generally, animals experienced little core temperature change throughout. Animal baseline core temperatures ranged from a minimum of 101.4 degrees Fahrenheit (°F) to a maximum of 105.4°F. Animals experienced increases of up to +1.7°F and decreases by -0.6°F with ultimate temperatures ranging from 104.4 to 107.8°F.

Three of the 18 animals exposed to HEMI devices died during the experiment as shown in Table 1. Two of the animals were exposed to the TASER®C2 and one was exposed to the Stinger™S-200. Animal 8 died four minutes after the second stimulus while animals 11 and 17 died six and five minutes after the first stimulus, respectively.

Catecholamine, cortisol, and lactate levels are shown in Figures 1 and 2. There was no statistical difference in blood gas parameters between pigs that died and pigs that survived. All pigs showed a mixed metabolic and respiratory acidosis.

There was large individual variability in catecholamine (epinephrine, norepinephrine, and dopamine) levels at the six measured time points. Because of this variability, the differences

between the groups did not reach statistical significance among devices or between survivors and non-survivors. Figures 1a, 1b, and 1c show group (device type) average data for the three catecholamines measured. Animals that died during the experiment are showed in separate lines from the group. The Stinger™S-200 device tended to elicit lower epinephrine and norepinephrine levels especially following the three minute exposure. The highest observed values in dopamine and norepinephrine were elicited by the TASER®X26, while the highest levels of epinephrine were elicited by the TASER®C2. Two of the animals that expired, animals 11 and 18 had marked increases in epinephrine following the first exposure and prior to death. Animal 8 had epinephrine values that nearly paralleled the group average, yet died following the second exposure. The three animals that died also tended to have high norepinephrine levels. The dopamine levels tended to be lower in the animals that died.

Among pigs exposed to the three different devices, cortisol levels tended to decrease following the initial 60 second exposure and remained steady or slightly increased over the one hour rest period indicating that there was little release of corticosteroids initially. During the second exposure, cortisol levels varied up and down, but there were no significant differences between groups for each device and no significant difference between those that survived and those that died. Since there was no significant difference in cortisol levels between devices, Figure 1d. shows cortisol levels grouped by animals that lived versus those that died.

Figure 2. shows lactate levels with group averages. The increases in lactate were smaller for the animals exposed to the Stinger™S-200, but the results were not statistically significant. Similarly, there were no clinically relevant or statistically significant differences between the animals that survived versus those that died in relation to any of the other biochemically

measured parameters (see methods), the monitored parameters (e.g. end tidal CO2, oxygen saturation, or EKG parameters such as S-T segment and T-wave changes.

There was an initial increase in blood pressure for all animals. Blood pressures were variable throughout application of the HEMI devices with no discernable pattern that could be associated with survival or death. There were no gradual increases or decreases in blood pressure or pulse volume/pressure. It was noted that the arterial blood pressure tracings showed a dramatic decrease in the blood pressure at the end of HEMI stimulation. Figure 4 is the arterial line tracing towards the end of the second (3 minute) HEMI stimulation shows the arterial line tracing recorded during the termination of the HEMI stimulation demonstrating the sudden decrease in blood pressure after HEMI simulation. While it was not possible to analyze the EKG during the HEMI device application, it was possible to discern the arterial wave from trace of the arterial blood pressure. At no stage were any extra-systoles noted during application of the HEMI device.

Animals that died were submitted for a complete necropsy conducted by veterinary pathologists. All three animals had degenerative cardiomyopathy with low vitamin E and selenium levels. Animal 17 was also diagnosed with severe pulmonary edema.

DISCUSSION

The observed motor response of animals to the devices was consistent from animal to animal for a particular device. Responses did differ depending upon the device. As noted earlier, while the Stinger™S-200 and TASER®X26 both resulted in tetany (sustained muscle contraction), the cyclic changes and intermittent low pulse repetition rate (8pps and 12pps) of the TASER®C2 resulted in observable severe and violent intermittent muscular responses by the animals. We sought to determine whether the sustained muscle contractions or the violent

intermittent responses cause the most stress; however, the stress hormone responses were not statistically significant between devices. The clinical observation that the Stinger™S-200 cause less intense muscle contractions could explain the clinically lower lactate levels observed in animals exposed to this device.

Visual observations made during HEMI application of the anesthetized animals showed more muscle movement (tonic and clonic muscle contractions) at a lower pulse wave rate than at a higher pulse wave rate. The TASER®C2 had a varying pulse wave rate, and at a low pulse wave rate, there were extreme muscle movements. When activated, the TASER®X26 caused complete sustained tonic muscle contractions throughout the body. The Stinger™S-200 had a constant pulse rate that resulted in relatively less extreme muscle contractions. It is likely that even though one device caused more extreme muscle movement, both devices caused maximum exercise of the muscles resulting in similar lactate production.

It appeared that the adrenal glands did not respond with a corticosteroid release to the initial stress of HEMI application either immediately following application or over a one hour rest period. It is possible that if swine are followed over a recovery period of 24 hours or more, one may see long term stress responses indicated by a change in serum cortisol levels.

Of particular interest was the immediate stress response in animals both during HEMI application and over a one hour rest period. Overall there was an increase in catecholamines during and immediately after HEMI application and a gradual decline over time after HEMI application. The increase in catecholamine levels showed an immediate sympathetic response to HEMI application, and despite a rest phase after an initial "stress event," the animal was able to mount a continued stress response during the second application indicating that there was not sympathetic exhaustion.

The three animals that died all expired several minutes after the application of the HEMI device had been discontinued. The pattern was that the blood pressure decreased over several minutes while the EKG demonstrated a normal sinus rhythm until pulseless electrical activity (PEA) developed. None of the animals developed ventricular fibrillation (VF) or ventricular tachycardia (VT) as a cause of death.

In some animals that survived, arterial blood pressure recordings at the end of the HEMI application showed a sudden decrease in blood pressure as soon as the HEMI device was stopped. This indicated one of two possibilities:

(1) The animals were hypovolemic and the peripheral vascular resistance was artificially maintained by the muscle contraction of the HEMI application. The stoppage of the HEMI "unmasked" the hypovolemia and the blood pressure dropped.

(2) The animals had normal blood volume with peripheral vascular resistance maintained by the HEMI (muscle contraction). When the HEMI application was ceased, the blood pressure dropped due to a sudden lower peripheral vascular resistance resulting from the increased pCO_2 and acidosis.

Additionally, if the animal started out hypovolemic, then one would expect a higher incidence of shock/death. When the oxygen partial pressure decreases to around 40 mmHg, there is a sudden massive outpouring of catecholamines. From clinical experience, it has been noticed that humans, at this stage, take a last gasp, and the rectal and urinary sphincters relax. In the case of animals, this massive cathecholamine surge might be sufficient to overcome the decreased peripheral vascular resistance.

This would then mean that death or no death (or level of shock) is actually animal dependent (i.e. dependent upon the state of the physiology of the particular animal, not equipment or device dependent).

Furthermore, the animals that died also did not exhibit a higher than average catecholamine response, indicating that death is not associated with excessive catecholamines in our model.

All three animals that died had low levels of vitamin E and selenium and associated cardiomyopathy. Nutritional vitamin E and selenium deficiencies can result in nutritional myodegeneration of cardiac or skeletal muscle. The cardiac form of disease can result in peracute to acute myocardial decompensation. Animals can be clinically normal and have an acute sudden onset of illness (Smith, 2002). Because cardiomyopathy can be seen in the cardiac form of nutritional myodegeneration, the cardiomyopathy seen in these animals could not be determined to be caused by the HEMI devices or the nutritional deficiency. Because all 18 animals were clinically healthy and were obtained from the same source and were on the same diet, it is unlikely that the low vitamin E and selenium levels were relevant. A respiratory virus was diagnosed in animal 17 which could have led to the pulmonary edema and mild bronchopneumonia despite the animal being clinically healthy and all pre-procedural blood parameters (complete blood count and chemistry screen) were within normal limits. However, one cannot rule out exposure to an HEMI device as a possible factor.

It is likely that multiple seemingly minor disease processes could combine to lead to death. A possible weakness of the study is that our stress model did not adequately represent a chronic stress state, such as long-term exposure to cocaine where changes in body mass index

and heart weight could occur (Karch et al. 1998), and a future study might be necessary to explore such a model.

CONCLUSIONS

Animals will likely not die during the HEMI stimulus. Death is more likely to occur **after** HEMI application is discontinued when the blood pressure suddenly decreases. In humans, any hypovolemia and/or low peripheral vascular resistance may not be manifested while the human is lying down (horizontal). If the human is placed upright (standing or sitting), then hypovolemia and/or low peripheral vascular resistance will be revealed. If the subject's sympathetic tone is insufficient to compensate for the hypotension, then the subject will become light headed and may become unconscious. This potential for hypotension (e.g. vasodilation due to increased pCO_2 and acidosis) could exist for quite a while after the HEMI exposure. The greater duration of HEMI exposure, the greater the potential for "post-exposure hypotension" to occur. Future studies should be designed to explore causes of hypotension.

Based on the results presented here, it can be concluded that one can exclude exhaustion of the sympathetic system as a cause of death. Furthermore, our results indicate that excess sympathetic stimulation is an unlikely major cause of, or contributor to, death.

PRACTICAL APPLICATIONS

For healthy adult animals, despite prolonged muscular exertion and physiological stress caused by HEMI devices, the body should be able to mount an appropriate sympathetic response and recover normally. This does not account for individuals with underlying health condition, who are taking prescription medication, or under-the influence of other drugs/alcohol.

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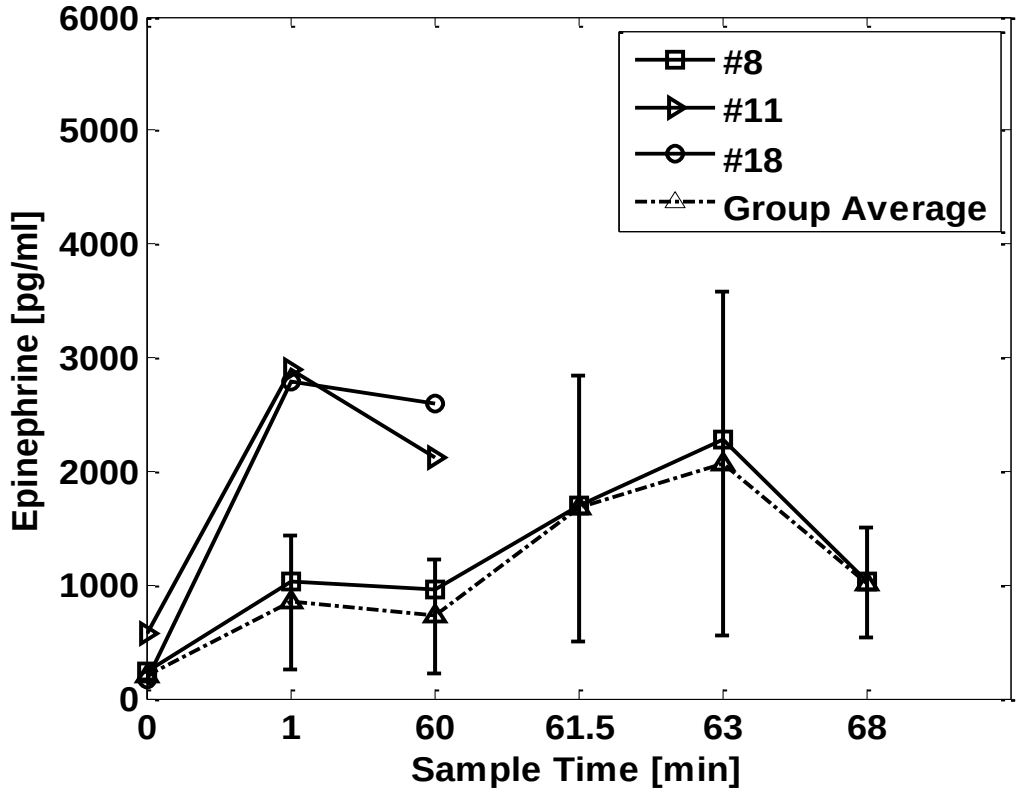
Figures

Figure 1 a., b., c., d. Epinephrine, Norepinephrine, Dopamine, and Cortisol levels of the group average of all pigs among three different HEMI exposures over time. Animals that died are shown in separate lines from the group average

1a.



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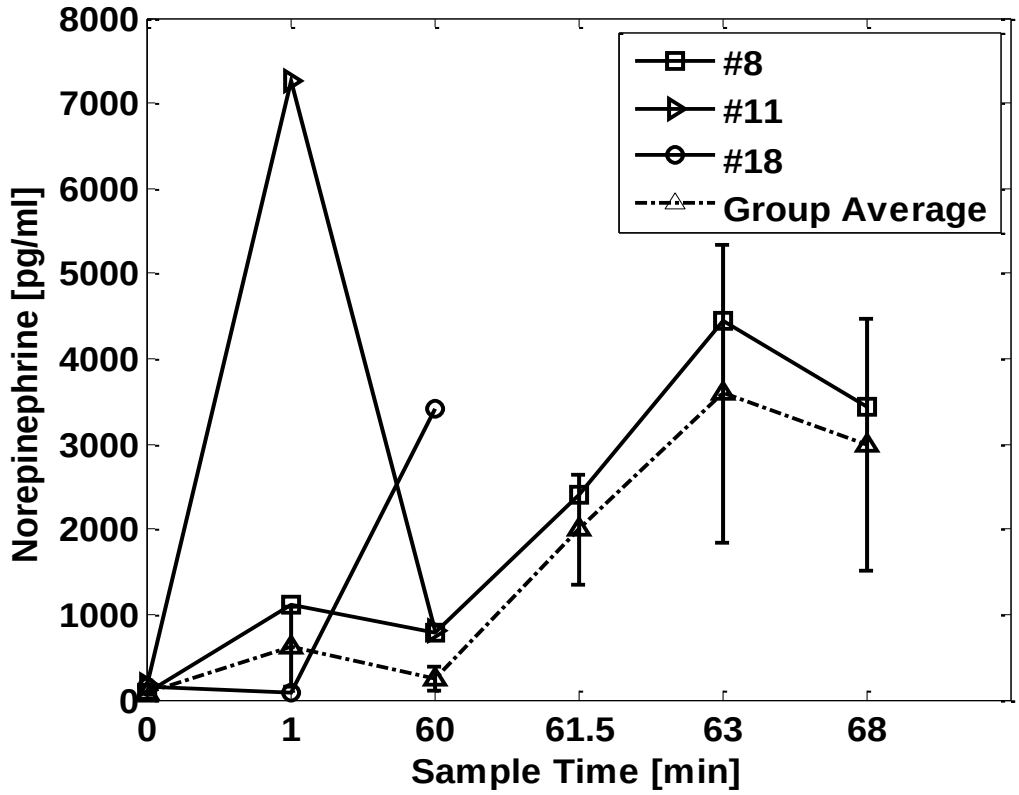
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4000
3000
2000
1000
0
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63
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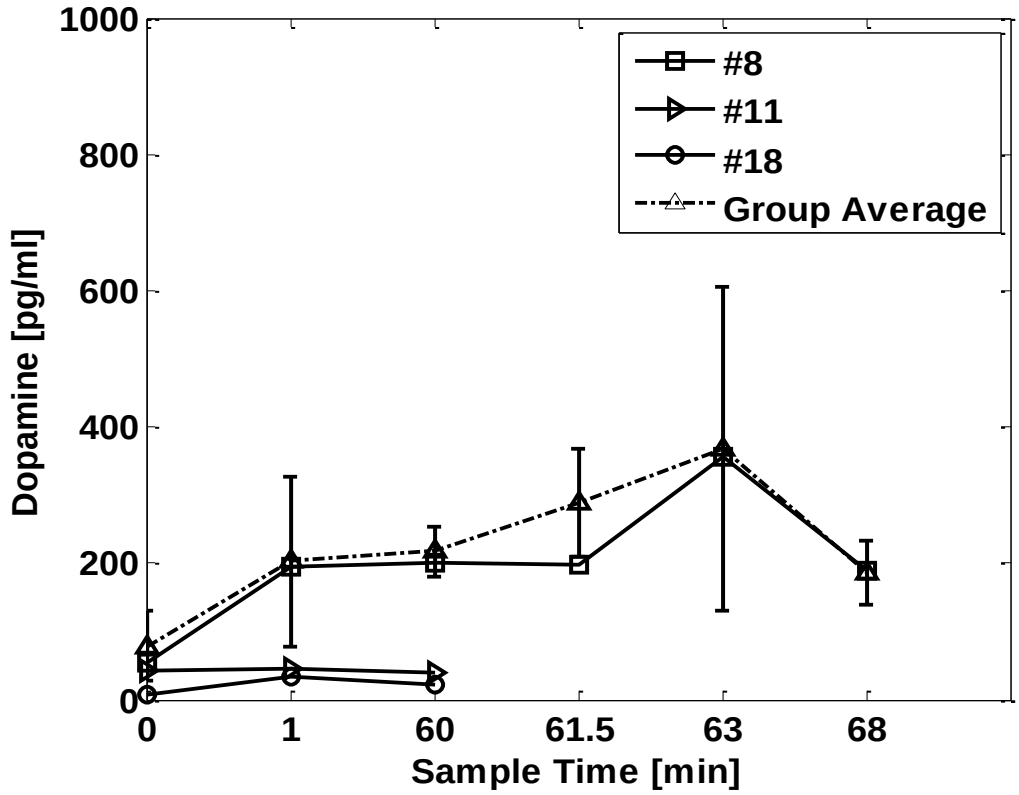
Sample Time [min]
#8
#11
#18
Group Average

1c.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Dopamine [pg/ml]
0
1
60
61.5
63
68
Sample Time [min]
#8
#11
#18
Group Average

1d.

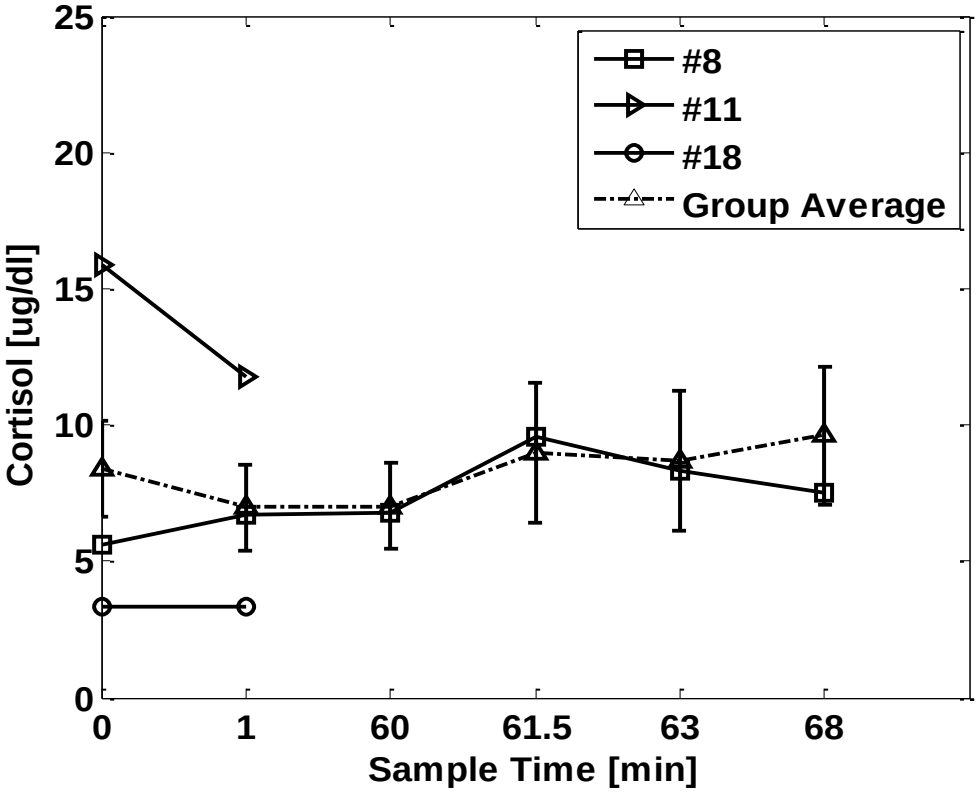
25
20
15
10
5
0
Cortisol [ug/dl]
0
1
60
61.5
63
68
Sample Time [min]
#8
#11
#18
Group Average

Figure 2. Lactate values during prolonged HEMI exposure showing average values at each time point for each device and for all devices together.